

The gut microbiome-derived metabolite butyrate is able to exert a protective action against the deleterious effects of ultra-processed foods in facilitating food allergy

L. Paparo^{1,2}, C. Bruno^{1,2}, E. Punzo^{1,2}, L. Voto^{1,2}, S. Coppola^{1,2}, A.F. de Giovanni Di Santa Caterina¹, A. Luzzetti^{1,2}, D. D'Ausilio^{1,2}, M. Lettieri¹, L. Carucci^{1,2}, R. Berni Canani^{1,2,3,4}

¹University of Naples "Federico II", Department of Translational Medical Science, Naples, Italy, ²University of Naples "Federico II", CEINGE Advanced Biotechnologies, Naples, Italy, ³University of Naples "Federico II", European Laboratory for the Investigation of Food-Induced Diseases, Naples, Italy, ⁴University of Naples "Federico II", Task Force on Microbiome Studies, Naples, Italy

Objectives and Study: Emerging data suggest a possible link between the consumption of ultra-processed foods, containing high level of advanced glycation end-products (AGEs), and the occurrence of food allergy (FA). It has been postulated that a healthy gut microbiome could exert a protective action against this effect. The short chain fatty acid butyrate is the most relevant gut microbiome metabolite able to modulate a broad range of protective actions against FA. We aimed to investigate the butyrate preventive action against the deleterious effects of AGEs in facilitating the occurrence of FA.

Methods: Human enterocytes (the Caco-2 cells) and peripheral blood mononuclear cells (PBMCs) from children at risk for atopy PBMCs (N=3, all Caucasian male, aged 48-60 months), were pre-treated with 1mM of butyrate for 24h and then, stimulated with 200 µg/mL AGE-Bovine Serum Albumin (BSA) or BSA, as control, for 48 hours. The effects on epithelial integrity (Trans-Epithelial Electric Resistance, TEER), permeability (tight junction protein occludin expression), pro-inflammatory cytokines (IL-8 and TNF-α) and Th2 (IL-4 and IL-5) release were assessed.

Results: Pre-incubation with butyrate resulted in a significant inhibition of the negative AGEs action on intestinal permeability (TEER and occludin expression reduction), and on the release of pro-inflammatory (IL-8 and TNF-α) and Th2 (IL-4 and IL-5) cytokines by Caco-2 cells and PBMCs, respectively.

Conclusions: These results support the importance of healthy gut microbiome in protecting against the negative effects elicited by AGAs in facilitating the occurrence of FA and support the role of butyrate as pivotal gut microbiome-derived metabolite for FA prevention.

Contact e-mail address: paparolorella@gmail.com